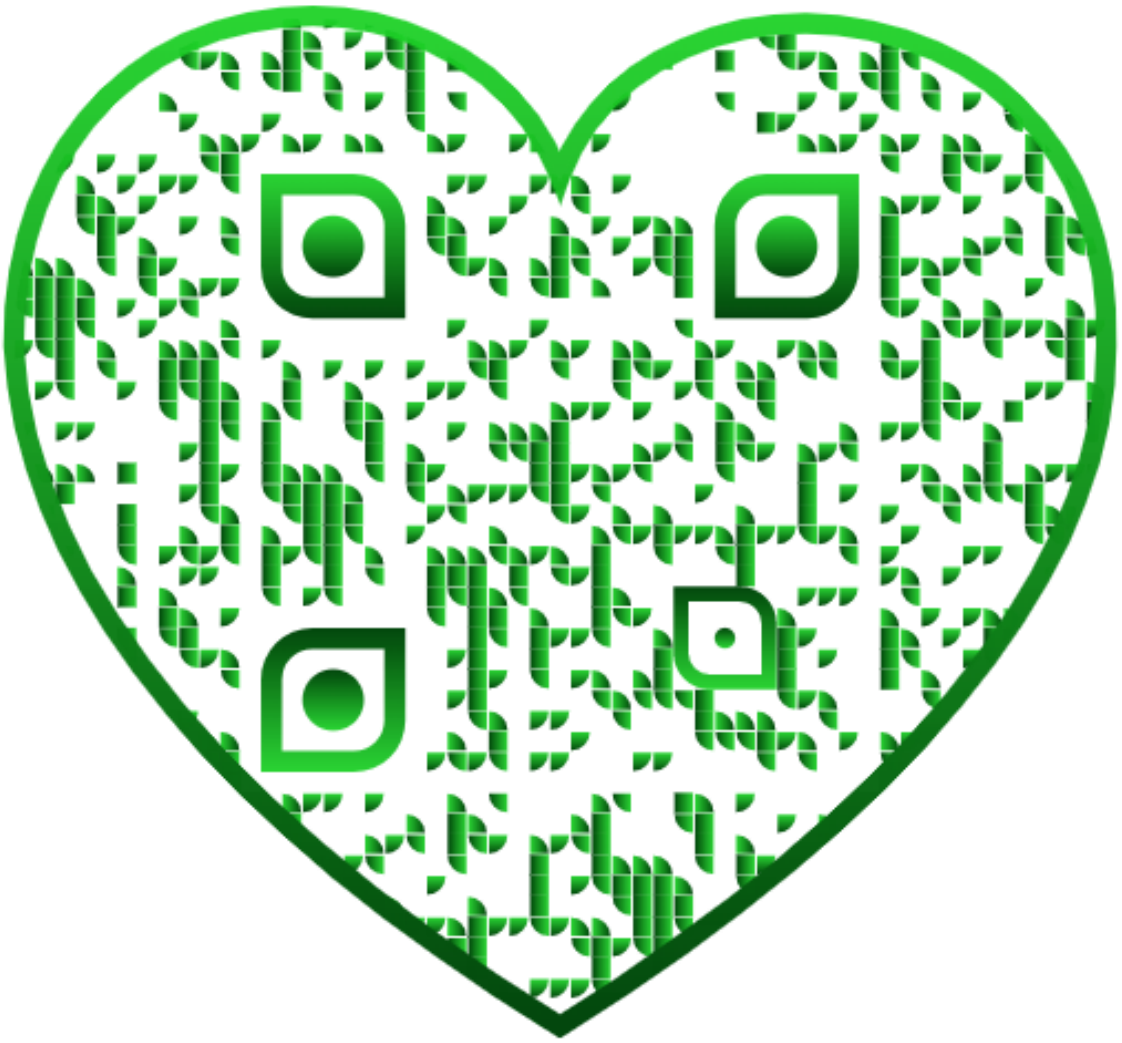
Q8

a. Provisional diagnosis ?

b. Causative organism? •

👉erysipelas

👉streptococcus pyogenes



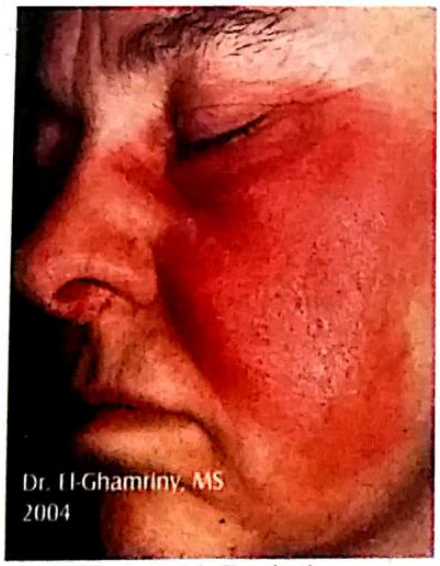


Photo 2-14: Erysipelas.

Erysipelas:

- A superficial form of cellulitis.
- It most often affects infants and the elderly, but can affect any age group.
- It is almost always caused by *Group A β-hemolytic streptococci*. Incubation period is 1-5 days.
- It predominantly affects the skin of the lower limbs, but when it involves the face it can have a characteristic butterfly distribution on the cheeks and bridge of the nose. Constitutional symptoms and fever are present.
- Affected skin is distinguished from other forms of cellulitis by a well-defined, raised border. The affected skin is red, firm, swollen and may be finely dimpled like an orange skin. Recurrence may induce lymphedema.

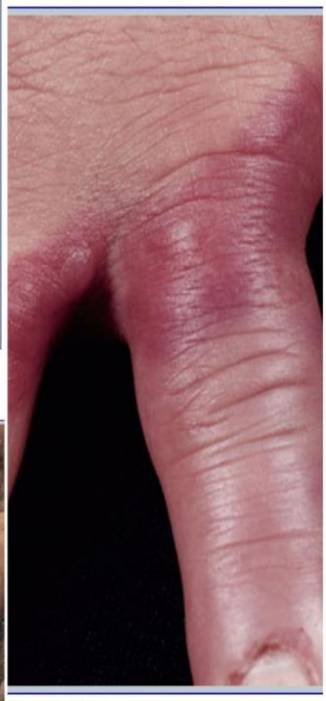
Complications of erysipelas:

1. Recurrent infections may result in lymphedema.
2. Nephritis, subacute abscess, and rarely septicemia.

Treatment of erysipelas:

1. Rest in bed and symptomatic treatment.
2. Systemic antibiotics; penicillins are the best; given for 10-14 days. In case of sensitivity to penicillin, macrolides antibiotics can be used.

Figure 25-29


re 25-31

Dignosis:-
- Erysipleas

Organism:-
- Streptococcus pyogenes.

Description:-
- well-defined area of hot, indurated, bright erythema that is painful and tender, there may be superimposed pustules, vesicles, bullae, or areas of hemorrhagic necrosis.

May be have scar

site... dermis and upper subcutaneous tissue

DDx:-
- cellulitis,
- erysipeloid breast cancer,
- irritant contact dermatitis,
- early necrotizing fasciitis or herpes zoster,
- erysipeloid,
- Sweet's syndrome

Treatment:-
- penicillin

the erysipelas different from cellulitis by well defined, red raised border

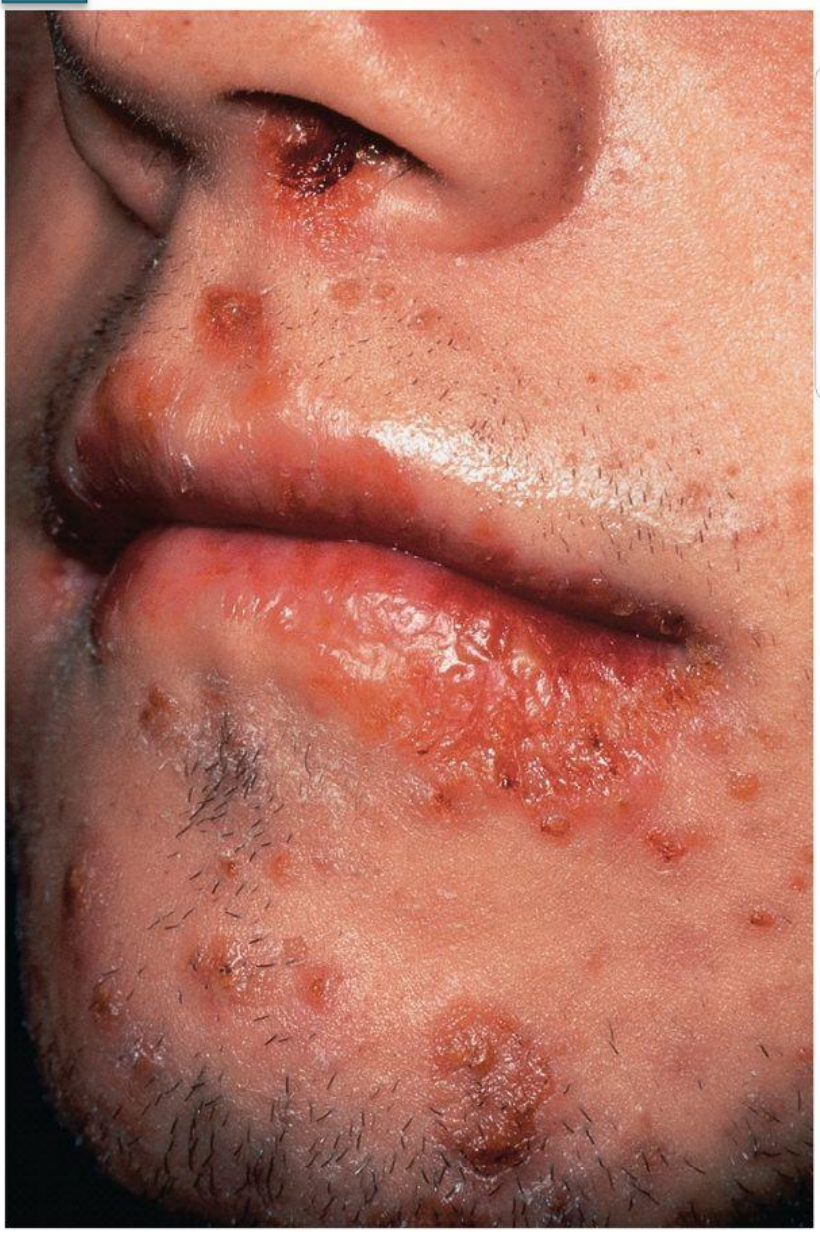


Fig. 29.12 Impetigo in a patient receiving isotretinoin. Multiple serous crusts are evident. Dryness and fragility of the skin, lips, and nasal mucosa in patients treated with isotretinoin increase susceptibility to staphylococcal infections.

Dignosis:-
impetigo
organism:-
staphylococcus&streptococucci

Description:-
- primary lesion (vesicles in erythematous base)
- secondary lesion (yellowish brown crust)

Predisposing factor:-
- -insect bites
- -pediculosis قمل
- -scabies
- -minor trauma

Site:-
- face around the nose&mouth, hand, neck,&extremitis

Healing:- without scar

Clinical type:-
1-non bullous impetigo
2-bullous impetigo
3-circinate impetigo

Complication:-
1-acute glomerulonephritis
2-spread of infection

DDx:-
1-tenia capitis
2-herpes simplex
Treatment:-
1-control predisposing factor
2-topical AB as fusidic acide&bacitracin
3-systemic AB as flucloxacillin or erythromycin

Q7

a. Provisional diagnosis ?

b. Write main lines of treatment ? •

👉impetigo

👉topical antibiotics:

as fusidic acid and bacitracin in mild and localized infection

Systemic antibiotics:

Flucloxacillin or erythromycin in sever and widspread infections

type of impetigo :-

1. Non-bullous impetigo....
*Thin walled vesicles on erythematous base > ruptured to form dry yellowish brown crusts > gradually peripheral extension without central healing
* no scar
*in severe case(present constitutional symptoms + adenitis)

2. Bullous impetigo...
* bullae lesion and less rapidly ruptured
* staph. Scalded skin syndrome
* with constitutional symptoms

3. Circinate impetigo...
* extension for ordinary or rupture of bullos

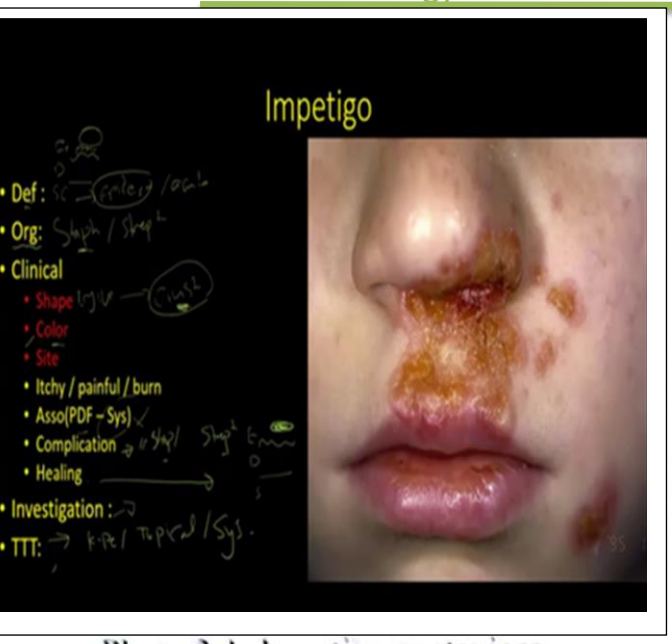


Photo 2-1: Impetigo contagiosa.

Photo 2-2: Impetigo contagiosa.
Superficial pustules and ulcers with yellowish crusting.

Photo 2-3: Impetigo of the scalp.

Impetigo contagiosa:

Impetigo is a highly contagious acute pyogenic skin infection common among children. It can appear anywhere on the body but usually attacks exposed areas.

Etiology:

Staphylococci and *streptococci*; commonly *Staph. aureus*, and *group A β-hemolytic streptococci.*

Predisposing factors:

- Overcrowded and unhygienic conditions.
- Humid hot weather.
- Itchy conditions, e.g. scabies & pediculosis.
- Malnutrition.

Mode of transmission:

- Physical contact with someone who has it.
- From sharing clothes, towels, etc.

Clinical types:

1. **Ordinary impetigo contagiosum:**
   - The most common type (about 70%).
   - Most common in children aged 5-10 years.
   - Typically affects the face, especially around the mouth and nose.
   - May occur on the extremities, genitalia, or the scalp (complicating scalp pediculosis).
   - Primary lesion is a thin-walled vesicle on an erythematous base. The vesicles rupture rapidly, that it is often not seen as such. There is a seropurulent discharge that dries to form golden yellow crusts.
   - No constitutional symptoms except fever in severe cases. Lesions often heal within 2 weeks (if untreated) leaving erythema that fades without scarring.
   - Regional lymphadenopathy is not uncommon.

Photo 2-4: Bullous impetigo.

Photo 2-5: Bullous impetigo in and immunocompromised adult. *Intact bullae can hardly be appreciated.*

2. **Bullous impetigo:**
   - An uncommon variant of impetigo contagiosa characterized by fragile fluid-filled or flaccid bullae (primary lesion is bulla surrounded by a rim of erythema). Central healing and peripheral extension may give rise to circinate lesions.
   - Almost always caused by pathogenic strains of *S. aureus* (Phage II type 71), which secrete toxins that cause acantholysis at the epidermal granular level.
   - Commonly affects neonates & infants whose kidneys cannot excrete the toxins.
   - The trunk is the site most commonly affected. May become generalized.
   - Regional lymph nodes usually are NOT enlarged.

3. **Circinate impetigo:**
   It results as an extension of impetigo lesions peripherally with central healing forming circinate or annular patterns or as a result of rupture of bullae in bullous impetigo.

Complications of impetigo (usually rare):

1. Spread of infection to other sites or other individuals.
2. Post-streptococcal glomerulonephritis (most serious) occurring in 2%-5% of cases 2-3 weeks following the acute infection.

Treatment of impetigo:

1. Treatment of any predisposing factor. If scabies and pediculosis are present, they should be treated as soon as impetigo is successfully treated.
2. Topical treatment:
   - Warm olive oil or potassium permanganate 1/8000 soaks to remove crusts.
   - Topical antibiotics, e.g. fusidic acid cream (Fucidin®), or mupirocin 2%.
3. Systemic antibiotic treatment, e.g. oral azithromycin (Zithromax®) for 5 days, or oral cephalexin (Keflex®) for 10 days, is indicated if infection is generalized, associated with fever or lymphadenopathy, and in case of bullous impetigo.

3

□Dx: Intertrigo

□Definition :
An inflammation of the flexural skin .

□Causes / DDx :
□infective :
* Streptococcal
*fungal : Candidial ,Tenia cruris
□Non-infective :
*simple friction
*seborrheic dermatitis
*psoriasis

Q9

a. Provisional diagnosis ?

b. Write main lines of treatment ? •

👉Candidal intertrigo
👉topical:
Nystatin
imidazole
tincture iodine
👉systemic :
Ketoconazole
fluconazole
or itraconazole
Also avoid humidity

INTERTRIGO

Definitions: intertrigo is inflammation of the flexural skin

Causes

A- Infective
- (1)streptococcal
- (2)fungal.
  - a-cndidial
  - b-tenia cruris
- (3) Erythrasma

B- Non- infective
- (1)simple (friction)
- (2)seb. dermatitis.
- (3)Psoriasis

Angular cheilitis

-It is the inflammation of the mouth angles

-Causes:
- (1) streptococcal
- (2) Candida
- (3) Vit. B deficiency

★ **Note**

Pseudo- folliculitis

It is the inflamation of skin of beard and results from re- entering of hairs to skin with shaving . →Irritation →papules.

Cutaneous involvement in systemic sterpt. Infections

1-Rash of scarlet fever.
2-Petechae, sub- ungual (splinter) haemoshages in subacute bacterial endocarditis .
3-Erythema nodosum .
4-Erythema multiform .

vitiligo :-Its an acquired loss of skin pigmentation

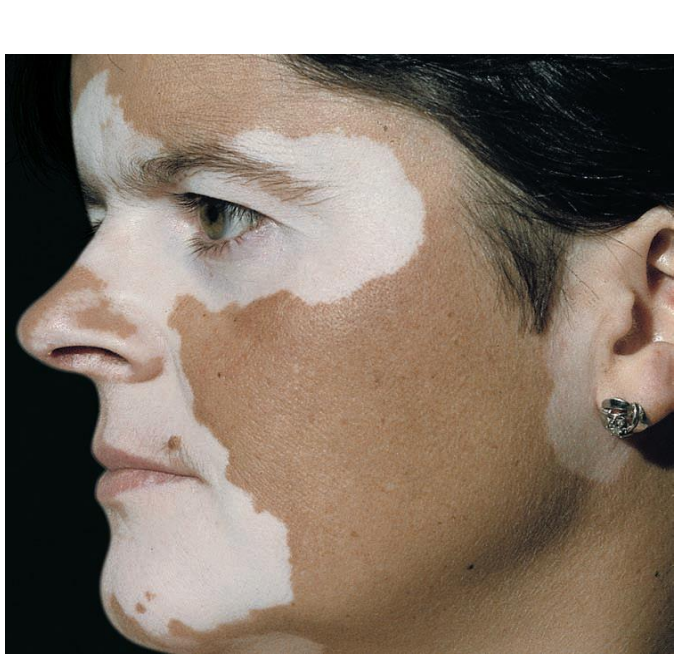
❄ Etiology ⇨ its ido pathical but may be due to
1-autoimmune 2-neurogenic 3-melanocyte self destruction

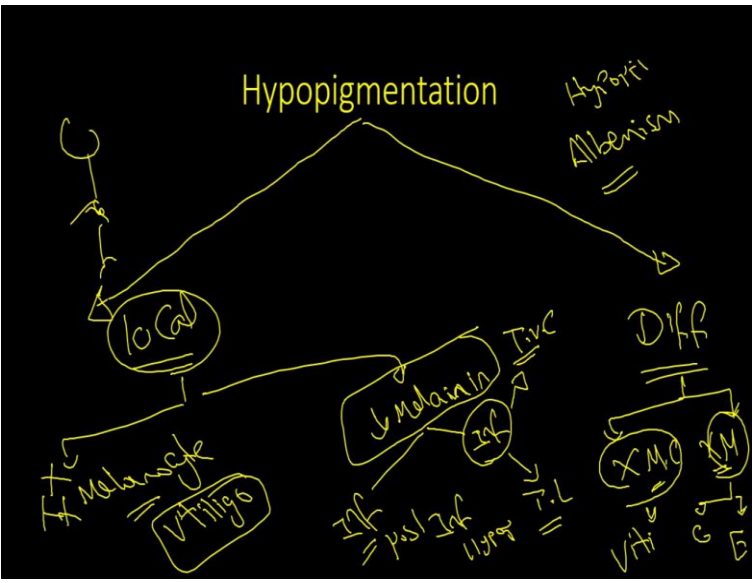
❄**Clinical picture:**
-DE pigmented white patches surrounded by normal or hyperpigmented skin

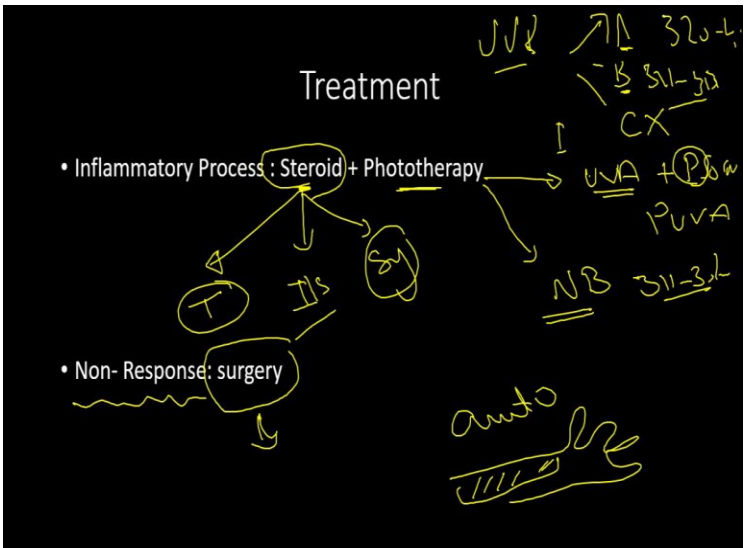
❄**Classification:**
-focal ⇨solitary or few scattered in 1 area
-segmental⇨unilateral macules in dermatomal distribution
-acrofacial ⇨ present in distal fingers or periorifacial
-Generalized ⇨ also termed vitiligo vulgaris and this is **the most commen type**
-mixed
-Universal ⇨present over most of the body

❄ . Diff D:
- postinfflamatory hypopigmentation ,pit alpa , pit versicolor ,

❄ ttt :
-phototherapy~ PUVA ,UVA
-medical ~steroid , immunomodulator ,antioxidant
-surgical
-depigmentation ~by monobenzyl ether if vitiligo present more than 60 % of body surface
-camouflage ~ skin colored material to hide affected skin







Q7

a. Provisional diagnosis ?

b.Types ? •

👉Vetilligo

👉localised :

- focal and
- segmental

👉generalised :

- acrofacial
- vitilligo vulgaris
- vitilligo universalis

٢٠٢٠/٨/٢٠ م

Designed by Khalil Siraj

Alopecia areata:-
Common asymptomatic disease characterized rapid onset of hair loss in sharply defined area

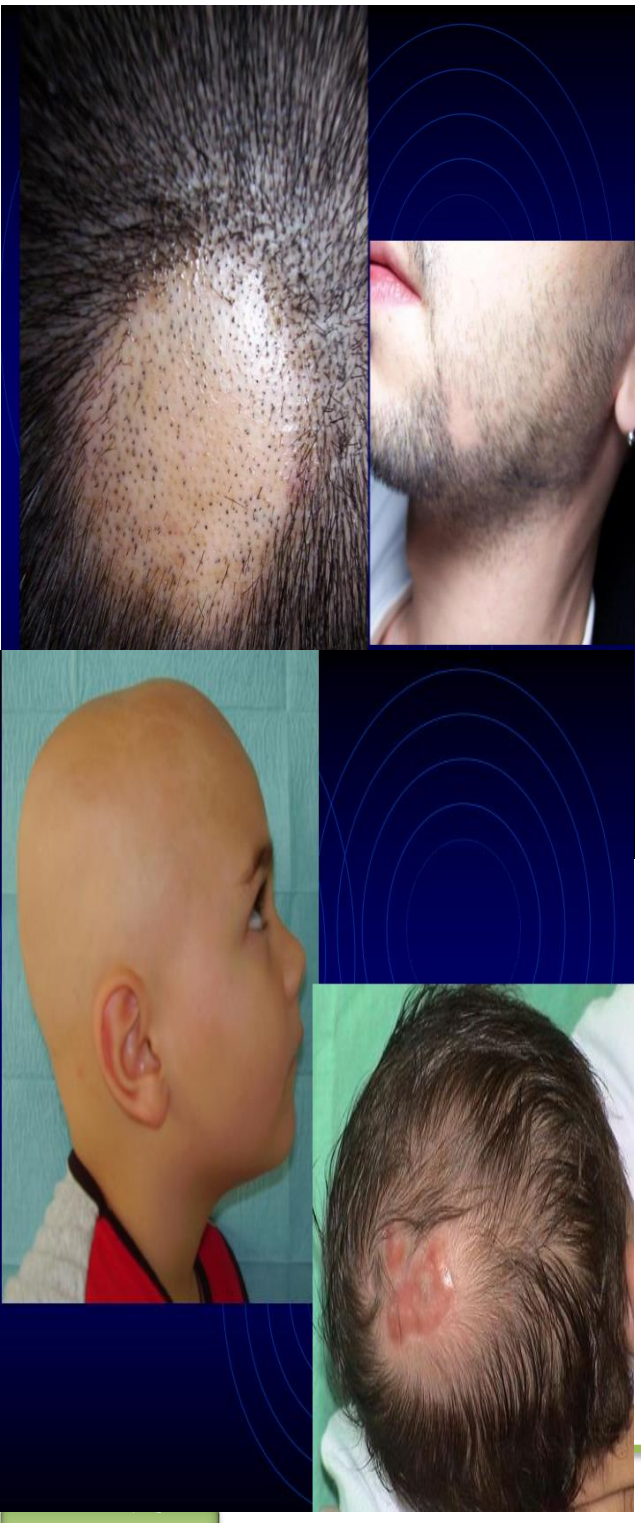
❄predisposing factors:
①genetic ②immunological ③emotional

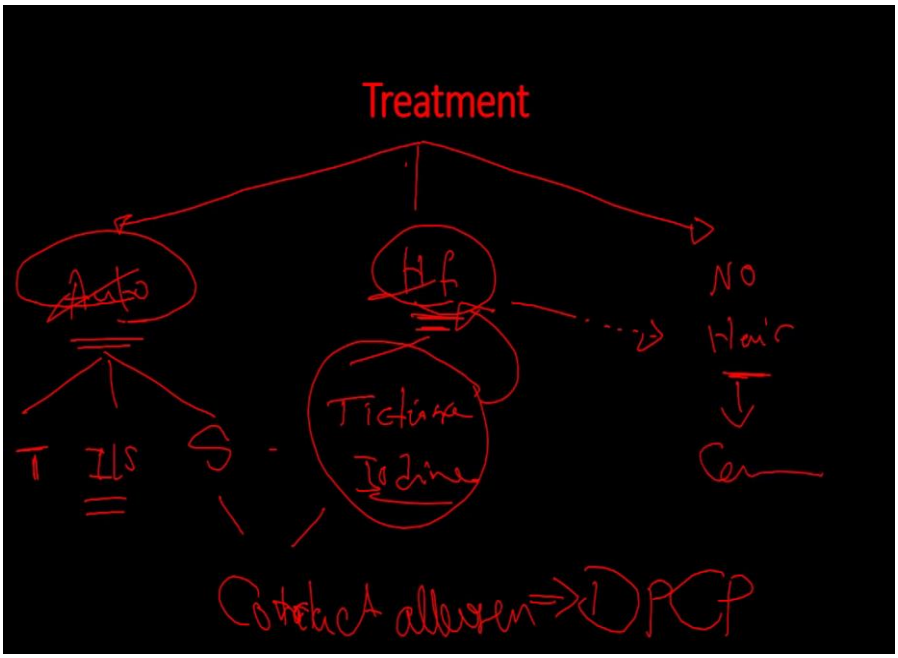
❄ clinical picture:
Rapid and complete loss of hair in one or more circumscribed round or oval patches.

❄Clinical types:
-localization
-totalis
-universalis

❄ ttt :
-Topical ~ steroid , local irritant , minoxidil , immunotherap, topical cyclosporine

-intacutaneous injection of steroid
-systemic ttt ~ steroid , photochemotherapy, systemic cyclosporine





ALOPECIA AREATA

Definition: It is a common skin disorder characterized by sudden loss of hair in a circumscribed area (or areas) usually on the scalp, but may involve other areas as the beard, moustache and eyebrows.

Etiology: is not settled. Many factors are implicated:

- Genetic factors: 10-20% of cases give positive family history.
- Immunological factors (most accepted): in favor of autoimmune mechanism.
- Psychological and emotional factors.
- Other factors: the condition may'follow reflex irritation of ocular or dental origin and focal sepsis.

Clinical picture:

- It can affect any hairy part of the skin.
- It presents as asymptomatic sudden onset of hair loss.
- The affected areas are variable in size.
- Single lesion mostly occurs but there may be multiple lesions as well.
- Characteristically, the scalp skin is completely normal with no crusts or scales.
- Loss of hair is complete with no hair roots or stumps.
- The presence of **exclamation mark hairs** (hairs which are thin proximally and thick distally) indicates progression of the disease.
- The appearance of thin, fine and almost colorless hairs within the patch of alopecia areata indicates starting re-growth.
- ***Clinical types:***
  1. *Patchy type:* the commonest.
  2. *Alopecia totalis:* the hair loss involves the whole scalp.
  3. *Alopecia universalis:* the hair loss involves the whole body surface.

Prepubertal onset, extensive involvement (especially on the posterior scalp) and atopy are signals of poor prognosis.

Differential Diagnosis: from other causes of localized non-cicatricial alopecia.

Treatment (depends on the extent):

1. Topical treatment:
   a. Primary local irritants such as tincture iodine, tincture capsicum and tincture cantharidis.
   b. Contact allergens such as diphenylcyclopropenone "DPCP".
   c. Corticosteroids: topical and intralesional.
2. Systemic treatment: with corticosteroids in extensive resistant cases.
3. Phototherapy: UVB or topical PUVA.
4. Reassurance and correction of any precipitating factors as error of refraction and septic foci.

Q6 •

a. Provisional diagnosis ? •

b. Write types? •

👉Allopecia areata

👉allopecia localisalis

Allopecia totalis

Allopecia universalis

Acne pustules

Closed comedon

lammatory nodule

Open comedones

✦**Acne vulgaris:**
Chronic inflammatory disease of pilosebaesous unit

✦**Pathogenesis**:↑androgen ⇨↑
①increase the sebum secretions ⇨↑keratine
②hyper keratosis
③proionibacterium acnes lysis triglycerides to free fatty acid

❍Primary lesion :polymorphic diseases (comedone:small punctate black lesion <black,white, papule ,pustule، nodule,cyst)

❍Post inflammatory lesion (erythema,pigmentation,scarring)

✦**classifications**:
①mild: comedone
②moderate: comedone,papule ,pustule
③sever :comedone,papule,pustule، nodule,cystic lesion

✦**Site** :
in **face** 99%then lesser **extent** of **back** 60%,**chest** 15%
In cheek Both sexes equally affected
in women ,3% in men(%١٢)

❍**Causes :**
①-Genetic factor
②Ductal hypercorification
③androgenic stimulation
④-presence of abnormal microbial flora(propionibacterium acne's)
⑤drug induced

✦ D.D :
*acne form eruption
*Rosacea
*perioral dermatitis

Q10

a. Provisional diagnosis ?

b. Type of lesions? •

👉Acne vulgaris

👉polymorphic :black head comedone, white head comedone, macules, papules ,pustules, eryhema

٢٠٢٠/٠٨/٠٩

Acne Vulgaris

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous units.

Pathogenesis

- Acne vulgaris is exclusively a follicular disease, with comedo formation produced by the impaction and distension of the follicles with tightly packed horny cells. Disruption of the follicular epithelium permits discharge of the follicular contents into the dermis. This, in turn, causes the formation of inflammatory papules, pustules, nodules and cysts.

Etiology Acne vulgaris is a multifactorial disease.

- **Genetic factors.** Several members of the same family may be affected.
- **Androgenic stimulation of sebaceous glands** with increased sebum.
- **Ductal hypercornification.** Comedones represent the retention of keratinocytes in the duct.
- **Presence of abnormal microbial flora** (Propionibacterium acnes).
- **Mediation of inflammation.** The dermal inflammation is not caused by bacteria but it probably results from biologically active mediators that diffuse from the follicle where they are produced by Propionibacterium acnes.

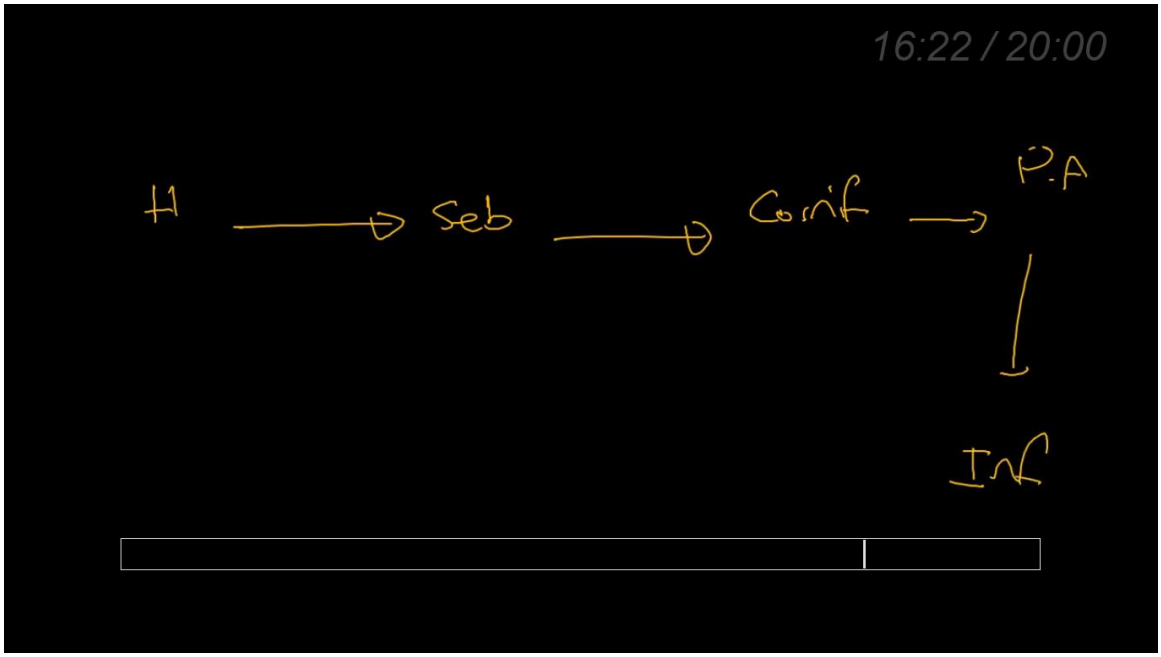
Clinical picture

Acne vulgaris is a disease of adolescents and young adults and usually disappears by the age of 25 years. Both sexes are equally affected.

- It occurs predominantly on the face (99%) and, to lesser extent, on the back (60%) and chest (15%). On the face it occurs most frequently on the cheeks, and to a lesser degree on the forehead, nose and chin. Acne is a polymorphic disease, but one type of the following lesions may predominate
- **Comedones (blackheads).** Comedo is the basic lesion in acne.It is a small punctate black lesion. When a comedo is extracted outside the follicular canal it appears as a yellowish worm-like structure with an upper black end (the black color is due to melanin not dirt).
- **Papules.** Small red papules.
- **Pustules.** Conical pustules.
- **Nodules and cysts.** Nodules, cysts, grouped comedones and scarring are features of acne conglobata which is a chronic disorder that may last up to 40-50 years of age.
- **Post-inflammatory lesions.** As erythema, pigmentation and scarring (small atrophic scars or pits, deeper furrows and keloidal scars).

Factors which may aggravate acne

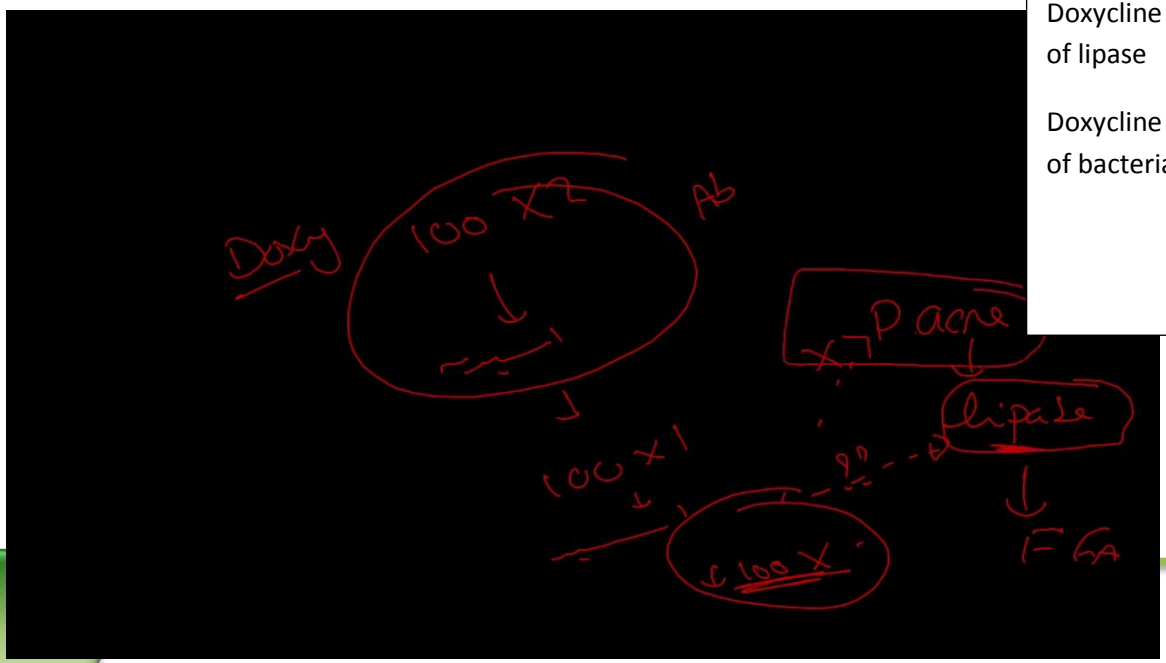
- **Menstruation.** About 70% of women complain of a flare 2-7 days premenstrually.
- **Stress.** It is unlikely that stress alone induces acne lesions. However, acne itself induces stress, and "picking" of the spots will aggravate the appearance. This is particularly obvious in young females who present with acne excoriée.
- **Sweating.** Up to 15% of acne patients notice that sweating causes a deterioration in their acne.



Treatment

H → P → Co → Anl

	S. Hormone (Androgen)	S. Sebum	T. Cornification	T + S P.Acne	Inflamation
Treatment	**Antiandrogens(*females***	**Systemic retinoids (isotretinoin:**	Retinoids sulfur ppt salicylic acid azaleic acid	Tetracyclin Doxycycline Erythromycin, Clindamycin.	Retinoids
Indication	Female	severe nodulo-cystic acne	Mild to Moderate		
Side Effect		Skin: Dryness MM: Dryness Hair: Loss Ms: Pain Liver: Inc.Enzyme Lipid : Increase	Post acne scars: dermabrasion, chemical peeling or LASER resurfacing		
Contraindication		Pregnancy			

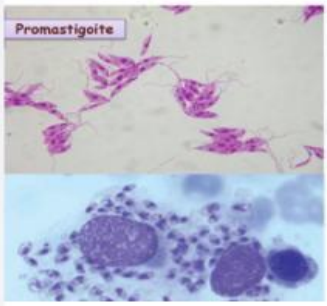


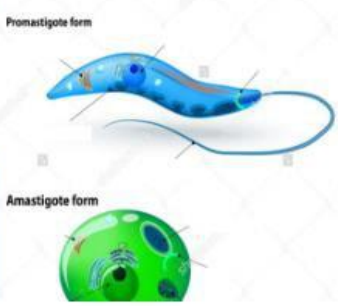
Doxycline loss dose → inhabtion of lipase

Doxycline large dose → inhbation of bacteria

D\D:
1- Cutaneous Leishmaniasis.
2- Ecthyma.
3- Malignant Ulcer.
4- Gumma (Tertiary Syphilis).
5- Lupus Vulgaris (TB).
Multiple, Different Size & Shape, Crusted Ulcerarative Nodules, on Erythematous Base Covered with Crust, Present on Right Forearm.

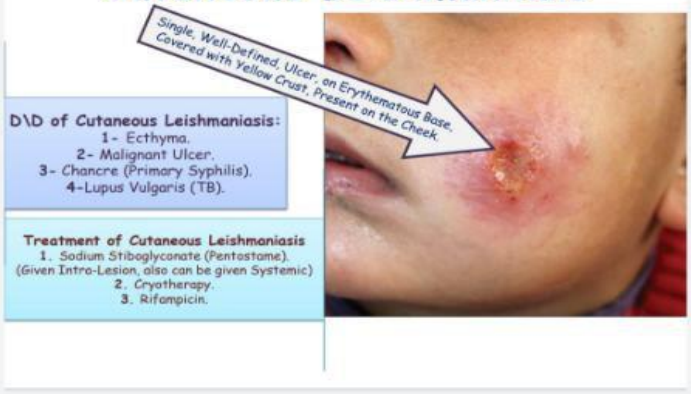
Amastigoite & Promastigoite



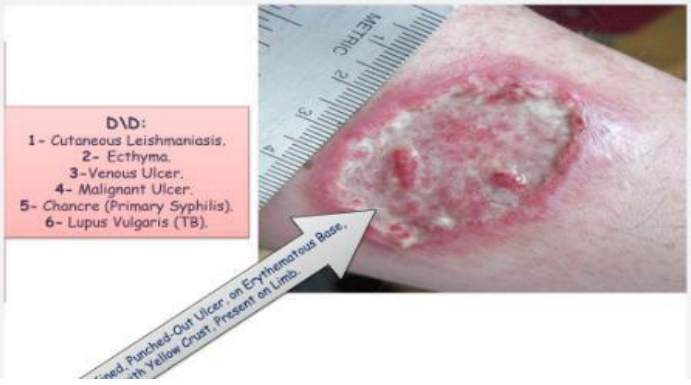


D\D:
1- Cutaneous Leishmaniasis.
2- Ecthyma.
3- Malignant Ulcer.
4- Gumma (Tertiary Syphilis).
5- Lupus Vulgaris (TB).
Multiple, Different Size & Shape, Crusted Ulcerative Nodules, on Erythematous Base, Covered with Crust, Present on Right Thigh.
08/10/2008

Cutaneous Leishmaniasis


Single, Well-Defined, Ulcer, on Erythematous Base, Covered with Yellow Crust, Present on the Cheek.
D\D of Cutaneous Leishmaniasis:
1- Ecthyma.
2- Malignant Ulcer.
3- Chancre (Primary Syphilis).
4-Lupus Vulgaris (TB).
Treatment of Cutaneous Leishmaniasis
1. Sodium Stiboglyconate (Pentostame).
(Given Intro-Lesion, also can be given Systemic)
2. Cryotherapy.
3. Rifampicin.

Multiple, Different Size & Shape, Well-Defined, Healed, Crusted Ulcers, Present on Extensor Surface of Left Forearm.
D\D:
1- Cutaneous Leishmaniasis.
2- Ecthyma.
3-Venous Ulcer.
4- Malignant Ulcer.
5- Gumma (Tertiary Syphilis).
6- Lupus Vulgaris (TB).



D\D:
1- Cutaneous Leishmaniasis.
2- Ecthyma.
3-Venous Ulcer.
4- Malignant Ulcer.
5- Chancre (Primary Syphilis).
6- Lupus Vulgaris (TB).
Single, Well-Defined, Punched-Out Ulcer, on Erythematous Base, Covered with Thick, Dark Crust, Present on Medial Surface of Left Leg.

✦ **Leishmaniasis**

- Leishmania Donovani / visceral leishmaniasis [kala azar]
- Leishmania Tropica / cutaneous leishmaniasis [oriental sore]
- Leishmania Braziliense / mucocutaneous leishmaniasis [Espundia]
- Cutaneous leishmaniasis

✨ **Etiology /**

- Leishmania major / wet [Rural] acute leishmaniasis
- Leishmania Tropica / dry [urban] subacute leishmaniasis

✨**mode of infection**

- bite of sandfly insect (the vector)
- direct contact with infected material

✨can effects any age and both sex on exposed parts

✨lesion / indurated nodules ~ ulcerates ~ ulcer with undermined edge surrounded by a red zone + 2ry infection may occur~ heal with big disfiguring scar

✨ **clinical types**

- acute cutaneous leishmaniasis
- chronic cutaneous leishmaniasis
- leishmaniasis recidivans
- Diffuse cutaneous leishmaniasis

✨ **Investigation**

- Skin smear / stained with geimsa stain
- Culture / on NNN media
- Biopsy / in chronic cases
- Immunological test / intradermal Leishmania test + serum examination for Leishmania antibodies

✨ **Diagnosis**

- History / endemic area + chronic skin lesion not respond to treatment
- clinical examination / characteristic lesion in exposed area
- investigation

✨ **Differential diagnosis**

- Ecthyma
- Furuncle
- lupus vulgaris

✨ **Management**

- prophylactic measures/ treatment patient in endemic areas + control of sandfly + avoiding the reservoir hosts + improve environmental sanitary
- local / excision of single lesion + cryotherapy with carbon dioxide , liquid nitrogen + intralesional injection of antimonial drugs + topical antibiotic as bacitracin or erythromycin
- systemic /★ pentavalent antimonial drugs [sodium stibogloconate] pentostam

★other drugs / Rifampicine _ Metronidazole _ ketoconazole_ intraconazole

Q5 •

a. Three differential diagnosis ? •

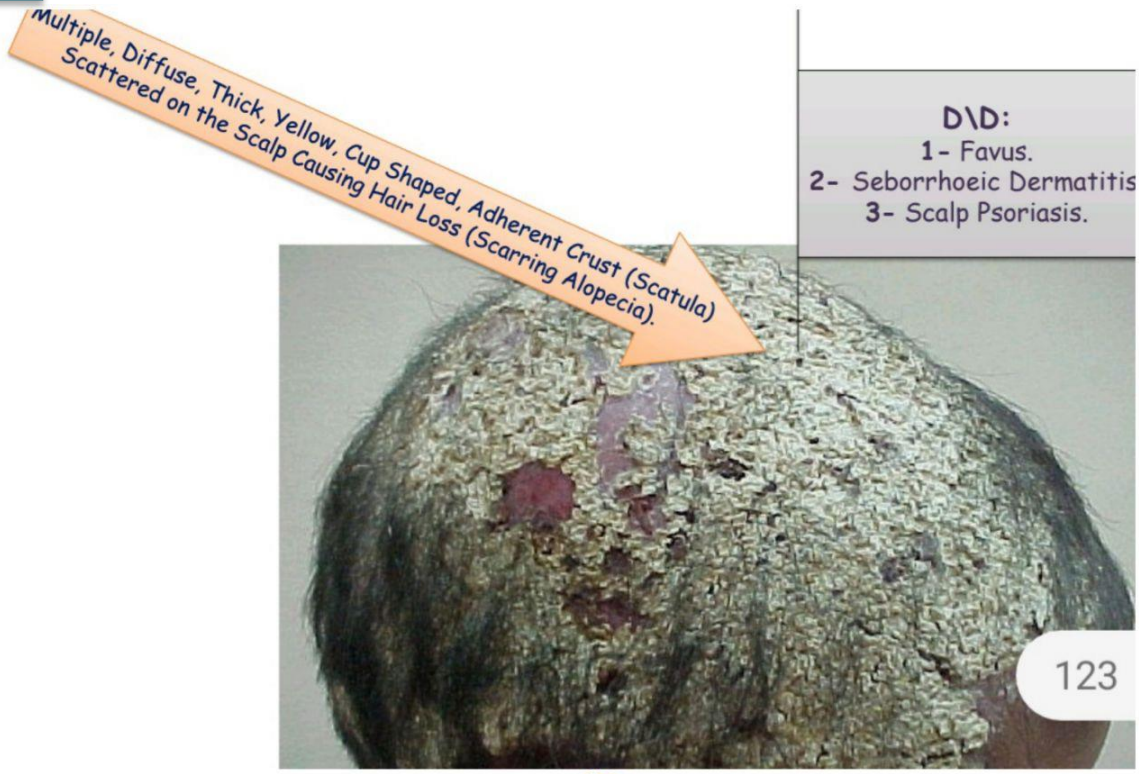
b. Write main investigations needed ? •

DD
cutaneous leishmaniasis
ecthyma
furuncle or carbuncle

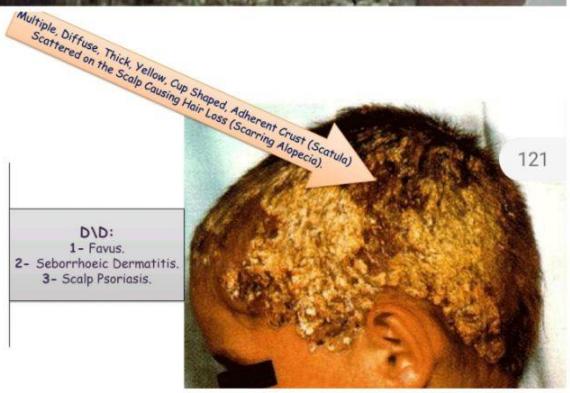
Investi:
To exclude cutaneous leishmaniasis
👉skin smear from periphery, stained by Geimsa stain and seen under light microscope revealing entra and extracellulat amastigoets
👉 biopsy if smear negative
👉culture on NNN medium reveal promastigoets
👉 leishmanin test (intradermal test)
👉serum examination for leishmania Abs

If these are negative
its not necessary to differentiate ecthyma from furuncle as their treatment is the same

٧-٨:٠ م


Multiple, Diffuse, Thick, Yellow, Cup Shaped, Adherent Crust (Scatula) Scattered on the Scalp Causing Hair Loss (Scarring Alopecia).
D\D:
1- Favus.
2- Seborrhoeic Dermatitis
3- Scalp Psoriasis.
123

Favus
118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D\D:
1- Favus.
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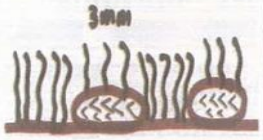
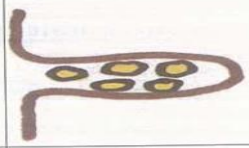
3- Scalp Psoriasis.
121

❖ Dermatophytes : = ring worm = tinea

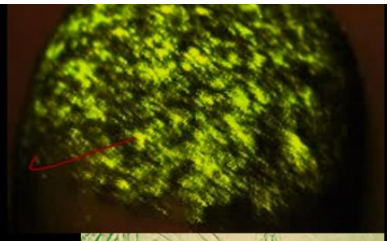
➢ **Clinical classification :**

1- Tinea capitis : = ringworm of scalp :

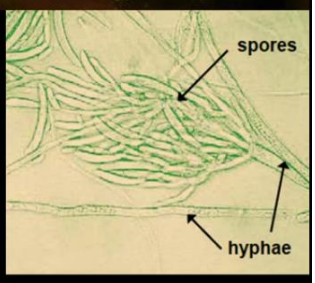
	Scaly type	Black- dot type	Kerion كرش	Favus فأر
				
Cause (MSK)	***Trichophyta*** Microspore	***Trichophyta***	***Trichophyta*** Microspore	***Trichophyta***
Epidemiology	*Children* only	*Children* only	*Children* and adults	*Children* and adults
Clinical picture	**S**ingle or multiple **bald patches** on the **S**calp, with fine **grayish white Scales** / *loose hairs* that break off → **S**tumps about 3 mm long that can be easily pulled out	Hairs break off at the surface of the skin giving picture of **black dot**	Boggy swelling studded with follicular pustules , pus is localized to hair follicles	Yellow cup-shaped sulfur crust (scutula) of mousy odor that form around *loose hairs* and lead to **diffuse** loss hair replaced by fibrous tissue
DD	**A**lopecia areata **P**soriasis **S**eborrheic dermatitis		**A**bscess	
Course	No scar	No scar	**Cicatricial alopecia (if not treated early)**	**Cicatricial alopecia**

	Black dot ringworm	Scaly ringworm	Favus	Kerion
• organism	T.violaceum	T.violaceum Microsporum canis	T.schoenleinii	severe inflammatory reaction T.verrucosum T.mentagrophytes
• hair & scales	single or multiple patches	similar to black dot	Diffuse > entire scalp	S & m swellings Hair easily detached few pustules
• Specific	with shortly cut hairs	More scales	thick yellow crust >cup shaped (sulfur cups or scutula)	painless
• Scaring	No	No	Yes	Yes
• LN	No	No	No	??

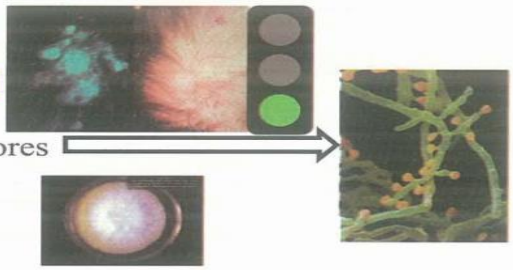
Investigation



- ***Wood's light examination:*** screening ?
  - M canis green fluoresce
  - T violaceum does not fluoresce
- ***Direct microscopy (KOH 10%)***
  - Hyphae
  - Spores
- ***Culture : Sabouraud's agar***
  - Macroscopic appearance
  - microscopic appearance



- **Diagnosis of Tinea capitis:**
  1. **Clinical examination**
  2. **Wood's light :** ***micros***pore gives **green fluorescence**
  3. **Direct *micros*copic examination** using 15 % potassium hydroxide to identify hyphae and spores
  4. **Culture** on Sabouraud's agar medium for *two weeks*
- **TTT of Tinea capitis :**



I- Topical therapy : USELESS IF USED ALONE

a. Tincture iodine 1- 2 % , Tolnaftate cream, whitfield ointment , azole derivatives.

b. Shampooing with *selenium sulphide* suspension or *ketoconazole* shampoo *twice weekly* renders the patient non- infectious early

II – Systemic therapy :

Griseofulvin, **12**.5 mg/kg/day after meals, for **6**- 8 weeks 2 divided doses.
(one tablet / 10 kg body weight with a maximum of **6** tablets)

- TTT: Gresiofulvin 12.5 Mg/Kg – 6-12 weeks
  - S/E: GID, photosensitivity, drug induced SLE.
  - #: pregnancy - hepatic failure - photosensitivity.

MCQ clinical

Q4 •

a. Provisional diagnosis ? •

b. Write main lines of treatment ? •

👉tinea capitus
👉👁topical :
Imidazoles :
as clotrimazole cream twice daily
and ketoconazole shampoo twice weakly
whitfield ointment twice daily
Allylamines as terbinafine

👁systemic :
Griseofulvin 12,5 mg/ kg at least 6 weeks
and in selected cases we can use itraconazole, fluconazole and terbinafine

م ٠٨:٢

Tinea capitis (scaly)

- lesion:circular scaly patch of hair loss
- DD:psoriasis- seborrheic dermatits- pityriasis rubra pilaris - tinea capitis
- no scarring
- affect children

tinea capitis (black-dot)

- lesion:patch of hair loss with black dot
- DD:alopecia areata-seborrheic dermatitis-
- no scarring
- affect children

Tinea capitis(kerion)

- caused by animal type of fungi
- Lesion:boggy indurated swelling with crusting and loose hair, follicle may be seen
- DD:pyogenic abscess-impetigo
- may followed by scarring and permanent alopecia
- occures in adult and children

✦②Atopic dermatitis :

Itchy ,chronic ,relapsing inflammatory disease

- (primary ,secondary lesion) :

Types of atopic :-

A/infantile phase (2m-2y) :

1/start in face but may be occur in any site

2/primary lesion :erythem and edematous papule

^_^ the papule are intensely itchy ,and may become exudative and crust .

B/Childhood (2-12y) :

1/Involved elbow and knee flexure,side of neck ,wrist and ankle

2/ description : Erythematous and edematous papules relapced by lichenification true eczematous with vesculation

^_^ Itching is intense .

C/ adult phase :(12y and more) :

Similar to childhood with lichenification

③/infective dermatitis:

Caused by micro organism or their product

□ Lesion : area of erythema with micro organism

Q1.

a . Provisional diagnosis ?/

b. Write classification/ types ?

👉Atopic dermatitis

👉- infantile phase :

From 2 months to 2 years

erythematus and odematus papules may become exudative and crusted

sites frequently start in the face and may occure anywhere

- childhood phase :

From 2 - 12 years

Erythematus and odematus papules replaced by lichenification

sites elbow and knee flexures, sides of the neck wrists and ankles

- adult phase :

More than 12 years

Lesions like childhood with pigmentation

sites like that of childhood

Q3 •

a. Provisional diagnosis ? •

b. Write main lines of treatment ? •

👉contact dermatitis

👉detection and avoidance of the cause

Use of Protective measures as gloves

Treatment of dermatitis :

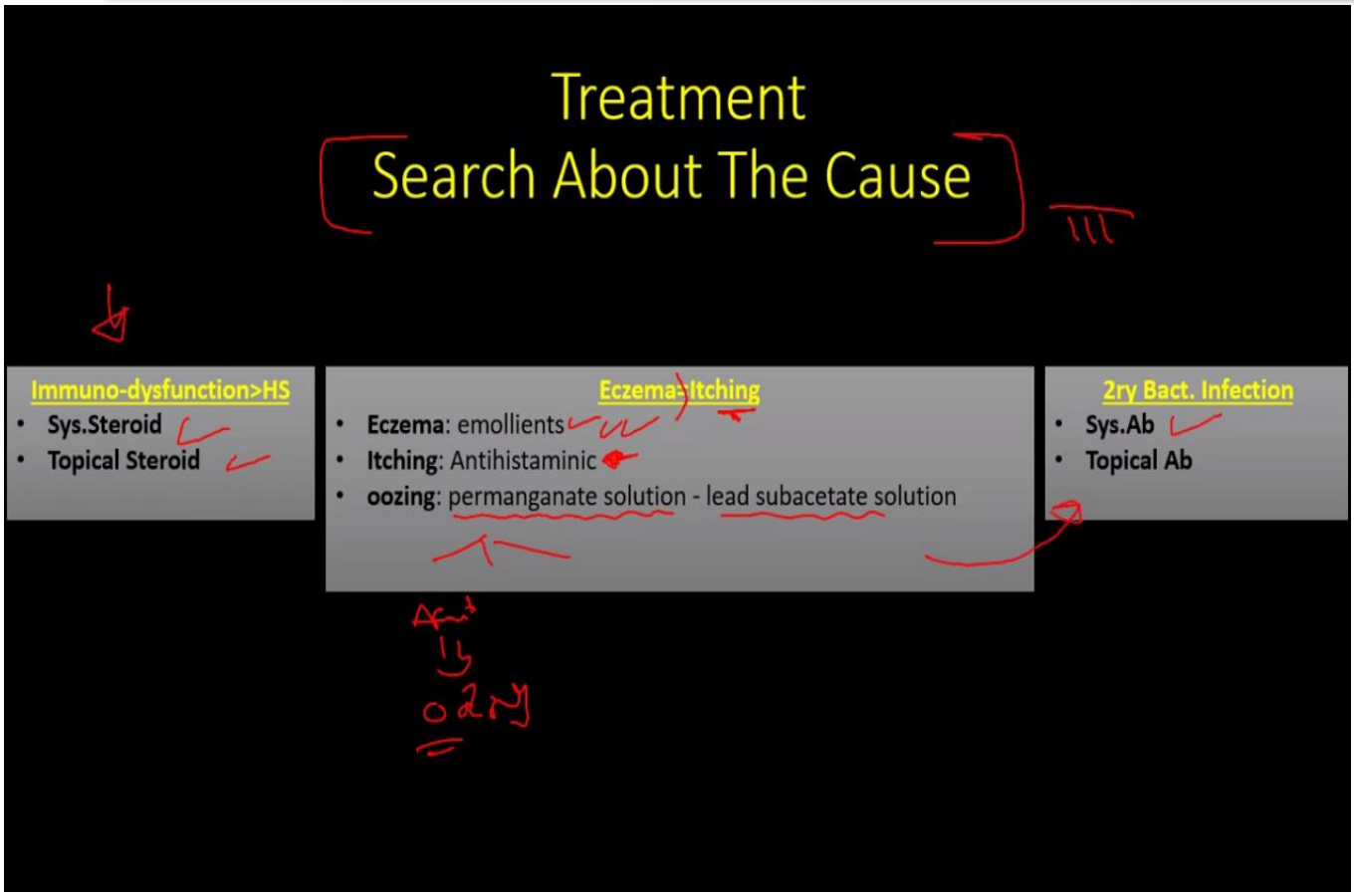
Emollients

antihistamine for itching

antibiotics local and sys for secondary bacterial infection

corticosteroids topical or systemic according to severity

Wet dressing as potassium permanganate for acute cases


Treatment
Search About The Cause
Immuno-dysfunction>HS
Sys.Steroid
Topical Steroid
Eczema-Itching
Eczema: emollients
Itching: Antihistaminic
oozing: permanganate solution - lead subacetate solution
2ry Bact. Infection
Sys.Ab
Topical Ab

Q2 •

a. Provisional diagnosis ?/ •

B. Write complications ? •

👉Herpes zoster (shingles)
👉post-herpetic neuralgia
ocular complications
fascial palsy
encephalitis
secondary bacterial infection and gangrene

Dx. Herpes zoster infection

Causative organism :
varicella zoster virus

MOT:
Reactivation of the virus in the sensory ganglion → migration of the virus into the skin

preciptating factors:
1- trauma
2- malignancy → lymphoma
3- drugs → cytotoxics and corticosteroids
4- low immunity

IP : 2-3 Weeks

the lesion usually appears unilateral as group of vesicles on an erythromatous base along the distribution of sensory nerve then become → pustules → crusts → healed with scars.

commonest sites:
Thoracic , cervical, trigeminal ,ophthalmic, and lumbosacral.

complication :
1- secondary bacterial infections.
2- scaring
3- motor paralysis
4- ocular zoster
5-facial palsy
6-pneumonitis
7- encephalitis
8-post-herpetic neuralgia

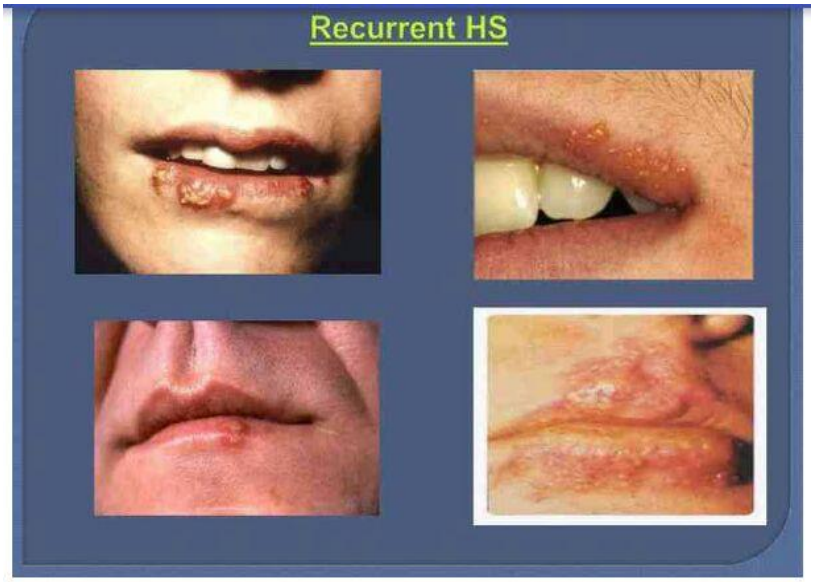
TTT:
TOPICAL :like HS
SYSTEMIC :
analgesic for pain → paracetamol
systemic corticosteroid→ hydrocortisone iv / prednisolone oral
systemic antibiotic → amoxicillin
systemic acyclovir + valacyclovir and famiciclovir in severe cases

1-Drying lotions as 5% **aluminium acetate** for the vesicular stage
2- antibiotic for secondary bacterial infection → erythromycine
3- acyclovir cream

DDx :
• Zosteriform HSV
• Localized contact dermatitis
• Bacterial skin infection (bullous impetigo, cellulitis

Dx. Chickenpox

Causative organism :
Varicella _ zoster virus
MOT: direct contact with lesions and droplet infection .
IP : 10_21 days
The lesions are polymorphic start as faint macules which rapidly develop into vesicles appearing in crops → umbillicated pustules → crusts on top of lesions
Site : centripetal → face ,trunk ,and oral mucosa
complications :
1- secondary bacterial infection
2-varicella pneumonia
3- keratitis
4-encephalitis
5-Reye syndrome
6-intrauterine / fetal varicella → zosteriform cutaneous scar ,limb hypoplasia, and eye damage .
7- neonatal varicella
Treatment :
Only symptomatic including
1- antipruritic → calamine lotion
2- antihistamine→loratidine
3- antibiotic for secondary infection →erythromycine
4- acyclovir for severe cases.
DDx :
Vesicular viral exanthems
• PLEVA
• Disseminated HSV
•Rickettsialpox
• Drug eruption
• Scabies
• Insect bites



- Dx: herpes simplex infection
- causative organism : Human herpes virus type I
- MOT :

Direct contact, droplets, or from infected secretions .

- IP : 2-12 days
- the lesions are small round vesicles on an erythematous base; often painful or burning; the grouping of these vesicles is a clue to the diagnosis; vesicles may become umbilicated or pustular, followed by erosions or ulcerations with hemorrhagic crusts, often with a scalloped border.
- **clinical types :**

1- herpetic gingivostomatitis
2- herpetic facialis / labialis
3- herpetic genitalis
4- keratoconjuctivitis
5-eczema herpeticum
6-neonatal herpes simplex

- triggering factors of recurrency :

•Trauma
•Immunodeficincy
•UV radiation
•Neural or dental surgery
•Emotional stress
•Menstruation

- **TTT:** TOPICAL :

1-Drying lotions as 5% aluminium acetate for the vesicular stage
2- antibiotic for secondary bacterial infection → erythromycine
3- acyclovir cream

SYSTEMIC :

For severe cases only oral acyclovir is the treatment of choice .

- DDx :

• perioral Impetigo
• perioral Coxsackie infection
• Aphthous stomatitis
•Drug-induced mucositis

 Dx: Molluscum contagiosum
causative organism :
Molluscum contagiosum virus → poxvirus
MOT :
Direct contact with infected person
the characteristic lesions are smooth pearly white,hemispherical,umbilicated papules, which on squeezing a white cheesy material is expressed.
site :
Multiple lesions occur mainly on the face ,trunk ,and extremities.
TTT :
•Spontaneous disappearance of the lesions may occur
•curettage and diathermy
•expression of the cotents of the lesion by squeezing with forceps
• salicylic acid 15-20%
•cryotherapy
• laser therapy
• KOH 5%
DDx:
•Papular ezcema
• juvenile xanthogranuloma
•Spitz nevus

Dx: warts/verrucae

causative organism :
Human papilloma virus

MOT :
1- inoculation to the skin either directly from infected person or indirectly from infected materials
2- Autoinoculation from one site to other in the infected persons.

predisposing factors:
Trauma and maceration with susceptible immunity

clinical types :
1-common warts → papules with rough horny surface.
2- plant warts →Smooth flat,or slightly elevated lesions ,skin colored or greyish ,the commonset site are face and back of hands.
3-filiform warts
4-digitiform warts
5-planter warts → sharpely defined , rounded, slightly elevated lesions with rough keratotic surface on the pressure zones of sole of the foots
6- anogenital warts

DDx:
seborrheic keratosis, actinic keratosis, cutaneous horn, trichilemmoma, Spitz nevus.

TTT:
Chemical ttt → salicylic acid
Electrocautery
Cryotherapy
Laser therapy

8. What are side effects of Corticosteroids ?

A- Side Effects Of Topical Corticosteroids :

- Skin atrophy
- Telanjiectasia
-Purpura
- hypertrichosis
- Secondary infection
- Systematic absorption Cushing's syndrome.

Incognito (tinea . scabies) steroid (acne - rosacea)

B- Side Effects Of Systemic Corticosteroids :

HTN
- Peptic ulcer
- DM - depression
- Psychosis
- Osteoporosis
- Pathological features
- Increasing susceptibility to infection -
- Cataract
- Skin thinning easy bruisin.

8. What are side effects of retetionod ?

✦teratogenicity in pregnancy اهم حاجة

① dryness skin

② dryness mucous membranes
③, muscle aches and pains,

④ photosensitivity,

⑤raised serum triglycerides or cholesterol,

⑥raised liver enzymes.

⑦ headache

⑦flushing are additional side effects observed with alitretinoin.

8. What are side effects of azoteroid ?

-tetra genic
-lipd profile ↑
-liver toxicity
-cns ⇨headache ,hair fall ,depreesion
-high cholesterol ⇨xantholsema
-noes⇨epistaxis
-dry
-bone ⇨ache bone

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- HTN
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- Psychosis
- Osteoporosis
- Pathological features
- Increasing susceptibility to infection
- Cataract
- Skin thinning easy bruisin.

9. Investigation should be done before giving Isotretinoin ?

- Liver Function tests.
- Lipid Profile.
- Pregnancy Test .

10. What are the side effects of Isotretinoin ?

- Tetratogenicity
- Hepatotoxicity
- Cheilitis
- Skin atrophy
- Hyperlipidemia
- Xerosis of skin
- Alopecia

نبض الحياة
Life Pulsation Batch
32

1- Enumerate primary and secondary skin lesions with definitions?

A. Primary lesions:

1- Macule: A circumscribed alteration in the color or texture of the skin less than 1 cm in diameter.
2- Patch: A macule lager than 1 cm in diameter.
3- Papule: A circumscribed palpable elevation less than 0.5 cm in diameter.
4- Nodule: A solid mass in the skin similar to the papule but more than 0.5 cm in diameter.
5- Plaque: An elevated area of skin 2 cm or more in diameter
6- Vesicle (blister): A fluid-filled lesion less than 0.5 cm in diameter. 7-
7- Bulla: A fluid-filled lesion larger than 0.5 cm in diameter.
8- Pustule: Small elevation of the skin similar to the vesicle but containing purulent material.
9- Wheal (weal): Evanescent, edematous, elevations of the skin of various sizes. It is the characteristic lesion of urticaria.
10- Comedo (blackhead): A plug of keratin and sebum in a dilated pilosebaceous orifice. Comedo is the characteristic lesion of acne vulgaris. .
11- Burrow: A small tunnel in the skin, linear, 'S' shaped, 3-5 mm in length and found in patients with scabies

B. Secondary lesions:

1- Scales: Dry or greasy laminated masses of keratin, may be primary in some diseases (e.g. psoriasis) or secondary (e.g. desquamation in scarlet fever). .
2- Crusts: Consists of dried serum and other exudates.
3- Erosion: A loss of epidermis which heals without scarring.
4- Ulcer: Complete loss of epidermis and dermis which heals with scaring.
5- 5-Fissure: A linear gap or slit in the skin surface.
6- Excoriations: Loss of skin substance caused by scratching with the fingernails to relieve itching.
7- Lichenification: A pattern of cutaneous response to repeated rubbing or scratching and characterized clinically by thickened skin, with accentuation of the surface markings .

2- What are different between vesicle and pustule?

- Vesicle (blister): A fluid-filled lesion less than 0.5 cm in diameter.
- Pustule: similar to the vesicle but containing purulent material.

3- What are types of warts?

1- Common warts
2- Plane warts (flat warts)
3- Filiform warts
4- Digitiform warts
5- Plantar warts
6- Anogenital warts (condylomata acuminata).

4- What are types of tinea according sites?

1- Tinea capitis
2- Tinea corporis or tinea cireinata
3- Tinea barbae
4- Tinea cruris
5- Tinea pedis
6- Onychomycosis (fungus infection of the nails).

☺ Investigation

سؤال اختبار عملي

1. Semen analysis
2. Urine & prostatic discharge analysis
3. Hormonal Profile (FSH, LH, T, PRL, others e.g.TSH)
4. Imaging (Scrotal US & Duplex, TRUS)
5. Testicular biopsy (???)
6. Other specific investigations (e.g., Antisperm antibodies, Electron microscopy, Sperm function tests, ...)
7. Chromosomal study (Karyotyping), Genetic study.

Semen analysis (WHO, 2010)

Parameter	Lower reference limit
Semen volume (ml)	1.5 (1.4–1.7)
Total sperm number (10^6 per ejaculate)	39 (33–46)
Sperm concentration (10^6 per ml)	15 (12–16)
Total motility (PR+NP, %)	40 (38–42)
Progressive motility (PR, %)	32 (31–34)
Vitality (live spermatozoa, %)	58 (55–63)
Sperm morphology (normal forms, %)	4 (3.0–4.0)
Other consensus threshold values	
pH	≥7.2
Peroxidase-positive leukocytes (10^6 per ml)	<1.0
MAR test (motile spermatozoa with bound particles, %)	<50
Immunobead test (motile spermatozoa with bound beads, %)	<50
Seminal zinc (µmol/ejaculate)	≥2.4
Seminal fructose (µmol/ejaculate)	≥13
Seminal neutral glucosidase (mU/ejaculate)	≥20

سؤال اختبار عملي

Cvp ,l,cmmv

* **Semen Analysis**
  - © **Color**: grayish-white
  - © **Volume**: 2ml or more (**60% seminal vesicles, 30% prostate & 10% epididymis & testis**).
  - © **PH**: 7.2 or more (alkaline)
  - © **Liquefaction time**: less than 30 minutes, **Viscosity**: Normal, how?
  - © **Sperm concentration/ml**: more than $20x10^6$/ml (& less than 250 millions/ml)
  - © **Sperm motility**: 50% or more of the spermatozoa show progressive motility (grades a & b) or 25% or more with rapid linear progression (grade a) wihin 60 minutes of ejaculation
  - © **Sperm morphology**: Normal forms at least 30% (WHO), or 14% (Kruger strict criteria).
  - © **Vitality**: 75% or more live. i.e. excluding dye.
  - © **WBCs**: fewer than 1 x 10^6/ml
  - © **Antisperm Antibodies**: fewer than 50% motile spermatozoa with beads (Immunobead test) or adherent particles (MAR test)
  - © **Biochemical markers (not routine):**
    - Fructose estimation (seminal vesicle marker): if bilateral congenital absence of the vas deferens is suspected.
    - L-Carnitine estimation (epididymal marker): may help differentiating pre from post-epididymal obstruction.

➢ **How to collect the sample ?????**
- If Normal semen analysis → no need to repeat it.
- If abnormal or Azoospermia → Repeat

➢ **Low semen volume or Normal volume????**

Semen Volume: (60% seminal vesicles, 30% prostate & 10% epididymis & testis):
- Short abstinent period (N= 2 - 7 days).
- Lost portion.
- Coitus interruptus.
- Idiopathic.
- Pathological causes (Hypogonadism, EDO, CAV, Retrograde ejac., Infection & fibrosis).
- Khat (Qat) chewing????

❍causes of male infertility (According to etiology):-

A. Pre testicular causes	B. Testicular causes	C. Post testicular causes
❍hormone 1-Hypothalamic disease() -Isolated LH deficiency ("fertile eunuch") -Isolated FSH deficiency -Gonadotropin deficiency (Kallmann syndrome) -Congenital hypogonadotropic syndromes 2-Pituitary disease -Pituitary insufficiency (tumor, radiation ,infection) -Hyperprolactinemia⇨↓FSH,LH 3-Exogenous hormones :- ♦↑estrogen ♦↑androgen ♦↑ glucocorticoid ♦excess, hyper- and hypothyroidism) -Growth hormone deficiency→FSH,LH→testiular development ❍systemic :- -Hepatic -Renal, Respiratory, Pancreatic. -Malignant	1.genetic ⇨Chromosomal ❖ Klinefelter syndrome ⇨extra X chrmoses ⇨XXY47 ❖ Down syndrome ❖ Myotonic dystrophy ❖ Noonan syndrome (46, XY) ❖ XX male (sex reversal syndrome) ❖ XYY male ❖ Mixed gonadal dysgenesis (45, X/46, XY) ❖ Y chromosome microdeletion syndrome ❖ Bilateral anorchia (vanishing testis syndrome) ②genetic :- -Cryptorchidism -Sperm disorders (Abnormal morphology; e.g.Globozoospermia, or ↓motiliy; e.g.Imm.cilia synd) ③Non chromosomal in nature Testicular origin: ❖ idiopathic ❖ acquired by ① Traumatic -Excess heat (occupation, sauna) -Testicular op. -Gonadotoxins :-(**drugs**(Chemotherapy), irradiation) ② Inflammatory ⇨mumps (Orchitis) ③ Neoplastic ④ Vascular -Varicocele -Testicular torsi -	1- Reproductive tract obstruction ❒Congenital blockages -Idiopathic epididymal obstruction -Congenital absence of the vas deferens (CAVD) -Young syndrome -Ejaculatory duct obstruction ❒Acquired blockages -Infection -Pharmacologic-alfa blockers -Vasectomy -Groin surgery -Functional blockages -Sympathetic nerve injury 2- Disorders of sperm function or motility -Infection -Immotile cilia syndromes -Maturation defects--imatur sperm -Immunologic infertility--ag, ab 3- Disorders of coitus Impotence Hypospadias—position of opening Timing and frequency

④Unexplained (Idiopathic) Infertility

- Genetic & chromosomal disorders (genetic Infertility) = 30% of sever Oligozoospermia and Azoospermia

Designec Khalil Si